REMARKS:

In the Office Action dated November 6, 2007, claims 1-5 and 10-11, in the above-identified U.S. patent application were rejected. Reconsideration of the rejections is respectfully requested in view of the following remarks. Claims 1-5 and 10-11 remain in this application, claims 6-9 and 12-20 have been withdrawn and new claim 21 has been added to the application.

Claims 1-5 were rejected under 35 USC §102(b) as anticipated by Apostolakis or Ginefri-Gayet. Applicants respectfully point out that Apostolakis discloses that MSO is a centrally acting neurotoxin with convulsive properties. Apostolakis also teaches that MSO can cause deformation, atrophy, loss of striation of muscle fibers, fibrosis and degeneration of Purkinje cells in the cerebellum. Apostolakis concludes that administration of MSO to rabbits in addition to the known convulsive effects may also be responsible for hind leg myopathy. The MSO dosage used by Apostolakis was 3-8 mg/kg body weight. Apostolakis does not suggest or disclose that MSO can be used to treat polyglutamine diseases such as Huntington's disease, spinocerebellar ataxia, and spinobulbar muscular atrophy and in view of the undesirable side effects discussed in Apostolakis (i.e. deformation, atrophy, loss of striation of muscle fibers, fibrosis and degeneration of Purkinje cells in the cerebellum) one skilled in the art would not be motivated to administer MSO to patients with a polyglutamine disease.

Ginefri-Gayet discloses that MSO, when administered at a convulsant dose (100-200 mg/kg body weight administered intraperitonealy or 50-75 µg per rat administered by ICV injection) induces a decrease in body temperature. Ginefri-Gayet indicates that MSO elicited a time dependent regional perturbation of 5-HT metabolism which could

be due to the marked rise in ammonia levels caused by the irreversible inhibition of the activity of glutamine synthetase. Ginefri-Gayet suggests that the 5-HT receptor plays a role in MSO elicited hypothermia in the rat. Ginefri-Gayet does not suggest or disclose that MSO can be used to treat polyglutamine diseases such as Huntington's disease, spinocerebellar ataxia, or spinobulbar muscular atrophy and in view of the undesirable side effect (hypothermia), one skilled in the art would not be motivated to treat patients with polyglutamine diseases with MSO in view of the disclosure in Ginefri-Gayet.

The applicant respectfully points out that the present claims are directed to a method for treating a polyglutamine disease which is not disclosed or suggested by either Apostolakis or Ginefri-Gayet. Therefore, applicants request that this rejection be withdrawn.

Claims 10 and 11 were rejected under 35 USC §102(b) as anticipated by Liedtke et al. Liedtke discloses an ion channel which is involved in osmoregulation and mechanoreception in vertebrates. The only mention of MSO in Liedtke is in paragraph 207 which discusses mammalian expression vectors such as a glutamine synthetase/ methionine sulfoximine co-amplification vector such as pEE14. There is no suggestion or disclosure regarding the administration of MSO for treating a polyglutamine disease. Since claims 10 and 11 depend directly or indirectly from claim 1 which recites a method for treating a polyglutamine disease, applicants request that this rejection be withdrawn.

Applicants respectfully submit that all of claims 1-21 are now in condition for allowance. If it is believed that the application is not in condition for allowance, it is

respectfully requested that the undersigned attorney be contacted at the telephone number below.

In the event this paper is not considered to be timely filed, the Applicant respectfully petitions for an appropriate extension of time. Any fee for such an extension together with any additional fees that may be due with respect to this paper, may be charged to Counsel's Deposit Account No. 02-2135.

Respectfully submitted,

By 

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